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NEWS 13  JUl 14  FSTA enhanced with Japanese patents
NEWS 14  JUl 19  Coverage of Research Disclosure reinstated in DWPI
NEWS 15  AUG 09  INSPEC enhanced with 1898-1968 archive
NEWS 16  AUG 28  ADISCTI Reloaded and Enhanced
NEWS 17  AUG 30  CA(SM)/CAplus(SM) Austrian patent law changes
NEWS 18  SEP 11  CA/CAplus enhanced with more pre-1907 records
NEWS 19  SEP 21  CA/CAplus fields enhanced with simultaneous left and right
                 truncation

NEWS EXPRESS  JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT
              MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
              AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

NEWS HOURS    STN Operating Hours Plus Help Desk Availability
NEWS LOGIN    Welcome Banner and News Items
NEWS IPC8     For general information regarding STN implementation of IPC 8
NEWS X25      X.25 communication option no longer available

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FILE 'HOME' ENTERED AT 08:34:54 ON 22 SEP 2006

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COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
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FILE LAST UPDATED: 21 Sep 2006  (20060921/ED)

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=> saha or trichostatin
          1314 SAHA
          1687 TRICHOSTATIN
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            10 TRICHOSTATINS
          1691 TRICHOSTATIN
                 (TRICHOSTATIN OR TRICHOSTATINS)
L1        2922 SAHA OR TRICHOSTATIN

=> leukotriene or lipoxygenase
         13967 LEUKOTRIENE
          7799 LEUKOTRIENES
         16083 LEUKOTRIENE
                 (LEUKOTRIENE OR LEUKOTRIENES)
         17010 LIPOXYGENASE
          1484 LIPOXYGENASES
         17245 LIPOXYGENASE
                 (LIPOXYGENASE OR LIPOXYGENASES)
L2       29136 LEUKOTRIENE OR LIPOXYGENASE

=> l1(l)l2
L3            5 L1(L)L2

=> d l3 1-5 ti

L3   ANSWER 1 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Responses to a histone deacetylase inhibitor, trichostatin A, in activated
     hepatic stellate cells of rats. Proteomic analysis for expression of
     altered target proteins

L3   ANSWER 2 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Short-chain fatty acids alter tight junction permeability in intestinal
     monolayer cells via lipoxygenase activation

L3   ANSWER 3 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Effect of eicosanoid formation on tight junction permeability changes in
     Caco-2 cells induced by short-chain fatty acids

L3   ANSWER 4 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Trichostatin A and structurally related histone deacetylase
     inhibitors induce 5-lipoxygenase promoter activity

L3   ANSWER 5 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Expression of 15-lipoxygenase-1 is regulated by histone acetylation in
     human colorectal carcinoma


=> d l3 4-5 ti fbib abs

L3   ANSWER 4 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Trichostatin A and structurally related histone deacetylase
     inhibitors induce 5-lipoxygenase promoter activity
AN   2003:466199  CAPLUS
DN   139:285631
TI   Trichostatin A and structurally related histone deacetylase
     inhibitors induce 5-lipoxygenase promoter activity
AU   Klan, Niko; Seuter, Sabine; Schnur, Nicole; Jung, Manfred; Steinhilber,
     Dieter
CS   Institute of Pharmaceutical Chemistry, University of Frankfurt,
     Frankfurt/Main, D-60439, Germany
SO   Biological Chemistry (2003), 384(5), 777-785
     CODEN: BICHF3; ISSN: 1431-6730
PB   Walter de Gruyter GmbH & Co. KG
DT   Journal
LA   English
AB   5-Lipoxygenase (5-LO) mRNA expression in Mono Mac 6 cells is
     induced by the histone deacetylase inhibitor trichostatin A
```

(TsA). In order to study the effects of TsA and several structurally related compds. such as MD85, D237 and M232 on 5-LO promoter activity, we have analyzed the response of a 5-lipoxygenase (5-LO) promoter luciferase reporter gene construct to histone deacetylase inhibitors in transiently transfected Mono Mac 6 and HeLa cells. We show that the activity of 5-LO promoter constructs comprising the sequences -778 to +53 and of several successive deletions of the 5-LO promoter is strongly increased upon TsA treatment. The data suggest a significant involvement of histone deacetylases in the regulation of 5-LO gene transcription. The basal activity of the 5-LO promoter strongly depends on the presence of multiple Sp1-binding sites (GC-boxes), five of which are positioned in tandem. Deletion of the five tandemized GC-boxes in the 5-LO reporter gene construct revealed that the induction of 5-LO promoter activity by TsA seems to be independent of these GC-boxes. Methylation of 5-LO reporter gene constructs by M.Hpall reduced 5-LO promoter activity but did not prevent induction of promoter activity by TsA, although the activated reporter gene activities were lower compared to the unmethylated plasmid, indicating the dominance of methylation over TsA-sensitive histone deacetylation in silencing of the 5-LO gene. The structure-activity data obtained for histone deacetylase inhibitors suggest that this assay system might serve as a cellular screening tool for the development of HDAC inhibitors.

RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
TI Expression of 15-lipoxygenase-1 is regulated by histone acetylation in human colorectal carcinoma
AN 2001:90389 CAPLUS
DN 134:264267
TI Expression of 15-lipoxygenase-1 is regulated by histone acetylation in human colorectal carcinoma
AU Kamitani, Hideki; Taniura, Seijiro; Ikawa, Hiroshi; Watanabe, Takashi; Kelavkar, Uddhav P.; Eling, Thomas E.
CS Laboratory of Molecular Carcinogenesis, National Institutes of Environmental Health Sciences, Research Triangle Park, NC, 27709, USA
SO Carcinogenesis (2001), 22(1), 187-191
CODEN: CRNGDP; ISSN: 0143-3334
PB Oxford University Press
DT Journal
LA English
AB 15-Lipoxygenase-1 (15-LO-1) is expressed at higher levels in human colorectal tumors compared with normal tissue. 15-LO-1 is expressed in cultured human colorectal cells, but only after treatment with sodium butyrate (NaBT), which also stimulates apoptosis and cell differentiation. The authors examined the regulation of 15-LO-1 in human tissue and the colorectal carcinoma cell lines Caco-2 and SW-480 by treatment with histone deacetylase (HDAC) inhibitors: NaBT, trichostatin A (TSA), and HC toxin. Northern and western anal. showed that expression of 15-LO-1 was up-regulated by these HDAC inhibitors. Furthermore, HDAC inhibitors stimulated promoter activity of the 15-LO-1 gene .apprx.12-21-fold using the -331/-23 region of the 15-LO-1 promoter, as measured with a luciferase-15-LO-1 promoter-reporter system, suggesting that 15-LO-1 is regulated at the transcriptional level by HDAC inhibitors. Histone proteins in colorectal cells were acetylated after treatment with HDAC inhibitors. Histone acetylation was also measured in human colorectal tissue and a correlation was observed between increased histone acetylation and 15-LO-1 expression. Thus, regulation of 15-LO-1 expression in colorectal tissues appears to occur by a novel and new mechanism associated with histone acetylation. Moreover, these results suggest that 15-LO-1 is a marker that reflects histone acetylation in colorectal carcinoma.

RE.CNT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

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=> asthma or brochitis or COPD
         32320 ASTHMA
            21 ASTHMAS
         32328 ASTHMA
                 (ASTHMA OR ASTHMAS)
             4 BROCHITIS
          2765 COPD
            15 COPDS
          2778 COPD
                 (COPD OR COPDS)
L4       34187 ASTHMA OR BROCHITIS OR COPD

=> d his

     (FILE 'HOME' ENTERED AT 08:34:54 ON 22 SEP 2006)

     FILE 'CAPLUS' ENTERED AT 08:35:21 ON 22 SEP 2006
L1        2922 SAHA OR TRICHOSTATIN
L2       29136 LEUKOTRIENE OR LIPOXYGENASE
L3           5 L1(L)L2
L4       34187 ASTHMA OR BROCHITIS OR COPD

=> l1(l)l4
L5           5 L1(L)L4

=> d l5 1-5 ti

L5   ANSWER 1 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Moraxella catarrhalis induces inflammatory response of bronchial
     epithelial cells via MAPK and NF-κB activation and histone
     deacetylase activity reduction

L5   ANSWER 2 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   NF-κB and Activator Protein 1 Response Elements and the Role of
     Histone Modifications in IL-1β-Induced TGF-β1 Gene Transcription

L5   ANSWER 3 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Repression of Interleukin-5 Transcription by the Glucocorticoid Receptor
     Targets GATA3 Signaling and Involves Histone Deacetylase Recruitment

L5   ANSWER 4 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Trichostatin A attenuates airway inflammation in mouse
     asthma model

L5   ANSWER 5 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Theophylline restores histone deacetylase activity and steroid responses
     in COPD macrophages


=> d l5 4 ti fbib abs

L5   ANSWER 4 OF 5  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Trichostatin A attenuates airway inflammation in mouse
     asthma model
AN   2005:164129  CAPLUS
DN   142:348684
TI   Trichostatin A attenuates airway inflammation in mouse
     asthma model
AU   Choi, J.-H.; Oh, S.-W.; Kang, M.-S.; Kwon, H. J.; Oh, G.-T.; Kim, D.-Y.
CS   Department of Veterinary Pathology, College of Veterinary Medicine and
```

School of Agricultural Biotechnology, Seoul National University, Seoul, S. Korea
SO Clinical and Experimental Allergy (2005), 35(1), 89-96
CODEN: CLEAEN; ISSN: 0954-7894
PB Blackwell Publishing Ltd.
DT Journal
LA English
AB Background Histone deacetylase (HDAC) inhibition has been demonstrated to change the expression of a restricted set of cellular genes. T cells are essential in the pathogenesis of allergen-induced airway inflammation. It was recently reported that treatment with HDAC inhibitors induces a T cell-suppressive effect. Objective The purpose of this study was to determine whether treatment with trichostatin A (TSA), a representative HDAC inhibitor, would reduce allergen-induced airway inflammation in a mouse asthma model. Methods BALB/c mice were i.p. sensitized to ovalbumin (OVA) and challenged with an aerosol of OVA. TSA (1 mg/kg body weight) was injected i.p. every 2 days beginning on day 1. Mouse lungs were assayed immunohistochem. for HDAC1, a major HDAC subtype, and for infiltration of CD4+ cells. The effect of TSA on airway hyper-responsiveness (AHR) was determined, and the bronchoalveolar lavage fluid (BALF) of these mice was assayed for the number and types of inflammatory cells, and for the concns. of IL-4, IL-5, and IgE. Results HDAC1 was localized within most airway cells and infiltrating inflammatory cells of asthmatic lungs. Treatment with TSA significantly attenuated AHR, as well as the nos. of eosinophils and lymphocytes in BALF. TSA also reduced infiltration of CD4+ and inflammatory cells and mucus occlusions in lung tissue, and decreased the concns. of IL-4, IL-5, and IgE in BALF. Conclusion TSA attenuated the development of allergic airway inflammation by decreasing expression of the Th2 cytokines, IL-4 and IL-5, and IgE, which resulted from reduced T cell infiltration. Our results suggest that HDAC inhibition may attenuate the development of asthma by a T cell suppressive effect.
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> logoff hold

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	28.85	29.06

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EAST Search History

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L3	0	L2 same L1	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/09/22 07:24
L4	12769	hydroxam$	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/09/22 07:24
L5	0	L2 same L4	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/09/22 07:24
L6	753894	transport	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/09/22 07:24
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L8	1648	hydroxyamide	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/09/22 07:24
L9	2	L7 near20 L8	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/09/22 07:24
L10	109	oxyamide	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/09/22 07:24
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EAST Search History

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EAST Search History

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L68	0	l64 same l65	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/09/22 08:01
L69	199	560/312.ccls.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/09/22 09:00
L70	1	l2 and l69	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/09/22 09:01